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EXAMINER	
JAISLE, CECILIA M	

ART UNIT	PAPER NUMBER
1624	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/587,836	MENGE ET AL.	
	Examiner	Art Unit	
	CECILIA M. JAISLE	1624	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *01 February 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-13,15,17 and 18* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-13,15,17 and 18* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *02-01-2008*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED OFFICE ACTION

Abstract

Applicant is reminded of the proper content of an Abstract of the Disclosure.

In chemical patent abstracts for compounds or compositions, the general nature of the compound or composition should be given as well as its use. As an aid to future researchers, a structural formula of the novel compounds should be given.

Lack of Unity

Applicant's confirmation of the election of Group I, claims 1-13, 15, 17 and 18 in the Response of Feb. 1, 2008 is acknowledged. With the current claim amendments, the claims are solely directed to the elected subject matter.

Rejection Under 35 US 112

Claims 17 and 18 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while enabling *in vitro* inhibition of PDE4BE activity, does not reasonably enable treatment of bronchial asthma, chronic obstructive pulmonary disease (COPD) or allergic rhinitis (claim 17) or psoriasis or atopic eczema (claim 18). The present specification offers no evidence that the claimed compounds control such specific diseases/conditions. The specification otherwise does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with claims 17 and 18.

Many factors require consideration when determining whether sufficient evidence supports a conclusion that a disclosure satisfies the enablement requirement and whether any necessary experimentation is "undue." MPEP 2164.01(a). These factors include: (1) the claim breadth; (2) the nature of the invention; (3) the state of the prior art; (4) the level of predictability in the art; (5) the amount of direction provided by the inventor; (6) the presence of working examples; and (7) the quantity of experimentation needed to use the invention based on the content of the disclosure. *In re Wands*, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)(reversing the PTO's determination that claims directed to methods for hepatitis B surface antigen detection did not satisfy the enablement requirement). See also *In re Goodman* 29 USPQ2d 2010, 2013 (Fed.Cir. 1993). Application of these factors to the present application supports the determination that the present disclosure fails to satisfy the enablement requirement:

1. **Breadth of the claims:**

(a) Scope of the compounds. Claim 30 covers potentially billions of compounds of Formula (I).

(b) Scope of the diseases covered. Claim 30 is directed to a method for treating COPD, RA and IBD, for which the disclosure is non-enabling.

COPD is a collection of progressive airway diseases, characterized by gradual lung function loss. It includes chronic obstructive bronchitis (inflammation and eventual scarring of bronchi) and emphysema (enlargement and destruction of alveoli). Emphysema comes in several forms, including congenital lobar emphysema, bullous emphysema, centrilobular emphysema (proximal acinar emphysema),

panacinar (panlobular), distal acinar (paraseptal) as well as Alpha-1 antitrypsin (AAT) deficiency, a genetic form of emphysema. COPD patients often have both bronchitis and emphysema. Ordinary chronic bronchitis is sometimes included with COPD even if there is no actual obstruction, and asthmatic bronchitis is generally included in COPD as well. Persons with COPD typically develop smaller air passageways, which can become clogged with mucus and have partially destroyed alveoli. There is no pharmaceutical treatment for COPD per se. Treatment is supportive and designed to relieve symptoms and improve quality of life. Oxygen is often given to partially compensate for the loss of lung function. Bronchodilators can expand passageways in the lungs, corticosteroids can reduce inflammation and antibiotics can ward off bacterial infections, but none of these treat COPD itself.

RA is an inflammatory disorder causing destruction of articular cartilage, in which macrophages accumulate in the rheumatoid synovial membrane. Mediators are cytokines, including IL-1, IL-18, α-TNF and IFN. It is thus an autoimmune condition where the body's immune system attacks its joints.

IBD is another illness considered to be associated with phosphodiesterase-4 (PDE4) activity. It is a generic term for an entire disorder family, the most important of which are ulcerative colitis and Crohn's disease. Less common forms include lymphocytic, collagenous, diversion, ischemic and infective colitis, radiation entero-colitis, solitary rectal ulcer syndrome (SRUS), antibiotic associated IBD, Behçet's Syndrome, and Infective Colitis. IBD has a range of known and unknown causes. Ulcerative colitis, Behçet's Syndrome and Crohn's disease, e.g., are idiopathic.

Partial tissue death (infarct) due to blood supply blockage, e.g. after major abdominal surgery or poor cardiac output in heart disease, can cause ischemic colitis. Cancer therapy can cause radiation enterocolitis. Infective colitis can arise from bacteria (e.g., shigella, salmonella, campylobacter, E. coli) or viruses (e.g., Norwalk-like virus rotavirus, CMV, HSV). Fecal stream diversion after ileostomy or colostomy can cause diversion colitis. Treatment depends on form, and some, e.g., radiation enterocolitis and SRUS, have no current effective pharmaceutical treatment.

2. **Nature of the invention and predictability in the art:** The invention is directed toward medicine and is therefore physiological in nature. It is well established that "the scope of enablement varies inversely with the degree of unpredictability of the factors involved," and physiological activity is generally considered to be an unpredictable factor. See *In re Fisher*, 166 USPQ 18, 24 (CCPA 1970).

Claim 30 is directed to therapeutic use of the present compounds in ameliorating COPD, RA and IBD related to PDE-4 inhibitory activity. Various PDE-4 types generally arise from presence or absence of two unique N-terminal domains called upstream conserved regions 1 and 2 (UCR1 and 2) and other pieces that may be present. UCR1 and UCR2 have been shown to form a module necessary for PDE-4 activation upon cAMP-dependent kinase (PKA) phosphorylation. For example, there are at least five different forms of PDE-4B: PDE-4B1, PDE-4B2 (short form), PDE-4B3, PDE-4B4 and very recently discovered, PDE-4B5. Distinct PDE-4A isoforms include PDE-4A1, PDE-4A5, PDE-4A4B, PDE-4A7, PDE-4A8, PDE-4A10 and PDE-4A11. PDE-4D has nine forms, 1-9. These various forms are not necessarily inter-

changeable and there is substantial variation in distribution even within the sub-families. Thus, PDE-4A1 is abundant in the brain, PDE-4A4B and PDE-4A10 in inflammatory cells, PDE-4A7 in the brain and spleen, and PDE-4A11 is widely distributed. The PDE-4D family is generally not seen in inflammatory cells at all. PDE-4D1 is seen in the spleen and heart, PDE-4D2 in the spleen, PDE-4D3 in brains, lung and kidney, PDE-4D4 and PDE-4D6 in the brain, PDE-4D5 in lung and kidney, PDE-4D7 in the brain and testes, PDE-4D8 in lung, heart and liver, and PDE-4D9 in spleen, heart and lung. Different types are regulated differently as well. ERK MAP kinases phosphorylate and regulate activity of PDE-4B, PDE-4C and PDE-4D but not PDE-4A isoforms. Reduced PDE-4D activity apparently causes defective RyR2-channel function associated with heart failure and arrhythmias. In dendritic cells (cells responsible for priming of naive T_h cells), PDE-4A is predominantly active, whereas monocytes mainly express PDE-4B. PDE-4D5 isoform preferentially interacts with signaling scaffold proteins, β-arrestin and RACK1. PDE-4D3 likewise forms a signaling complex with AKAPs such as AKAP450.

Pharmacological activity in general is unpredictable. In applications involving physiological activity, such as the present:

> The first paragraph of 35 U.S.C. §112 effectively requires that the scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art.

Plant Genetic Systems v. DeKalb Genetics, 65 USPQ2d 1452, 1456 (Fed.Cir. 2003).

3. **Direction and Guidance:** That provided in the specification is very limited. The dosage range information is meager at best. It is generic, the same for all disorders the specification covers. No specific direction or guidance provides a regimen or dosage effective specifically for conditions other than asthma and atopic dermatitis

4. **State of the prior art:** Sasaki, et al., Inflamm. Res., 53 (2004), 031-037, reported:

> In conclusion, we have demonstrated that the inhibitory effect of KF19514 on the TNF-alpha induced GM-CSF production by the BEAS-2B cells is mediated by the inhibition of the PDE4 activity and a cAMP-dependent pathway. PDE4 may be a possible target for the regulation of the cytokine production in epithelial cells. This may have implications of therapeutic value for the use of KF19514 and PDE4 inhibitors in inflammatory lung diseases including bronchial asthma.

A report from the European Respiratory Society, Feb. 13, 2007, http://www.newtocopd.com/currentaffairsnews/list751_item17680.aspx, downloaded Jan. 16, 2008, gives hope regarding the future of COPD pharmaceutical therapy: "Although there are currently no effective treatments for COPD, several new classes of anti-inflammatory drugs are now in clinical development and may be useful in treating the inflammation of COPD and chronic comorbid diseases."

Lipworth, Lancet, Vol. 365: Iss. 9454, 167-175, 2005, acknowledges the need for more research regarding PDE4 inhibitors for beneficial treatment of COPD:

> In COPD, the key study would be a long-term, placebo-controlled assessment over 1 year of a PDE4 inhibitor for its effects on exacerbations and quality of life, and subsequently over 3 years to look also at decline in FEV [forced expiratory volume], as has been done with high-dose inhaled corticosteroids. Ultimately, PDE4 inhibitors would have to be compared with other therapies such as long-acting anticholinergic drugs (e.g., tiotropium), combination

> inhalers (e.g., fluticasone/salmeterol or budesonide/formoterol), or theophylline, as recommended in current guidelines.

Barnes, Thorax, 2003: 58, pp. 803-808, cautiously states, regarding COPD therapy with PDE4 inhibitors (p. 805, col. 1):

> PDE4 is the predominant PDE expressed in neutrophils, CD8+ cells, and macrophages, suggesting that **PDE4 inhibitors might be effective in controlling inflammation in COPD**. Selective PDE4 inhibitors such as cilomilast and roflumilast are active in animal models of neutrophils inflammation. Cilomilast has some beneficial clinical effect in patients with COPD, and larger studies are currently underway. Roflumilast appears to be well tolerated at does that significantly inhibit TNF-alpha release from peripheral blood monocytes. **PDE4 inhibitors are limited by side effects,** particularly nausea and other gastrointestinal effects, **but it might be possible to develop isoenzyme subtype selective inhibitors in the future** which are less likely to be dose limited by adverse effects.

Dyke, Exp. Opin. Invest. Drugs (1999) 8(9), 1301-1325, noted the limited extent of COPD studies (p. 1309), "SB207499 is the only selective PDE4 inhibitor which has been evaluated in COPD patients."

Kawasaki, et al., Allergology International, Vol. 54, No. 3, 2005, pp. 427-433 (abst.), demonstrated a murine model of allergic nasal obstruction and cautiously suggested (p. 432), "PDE4 inhibitors may be useful for treatment of allergic rhinitis."

Regarding tests of PDE4 inhibitors in control of psoriasis and atopic dermatitis (atopic eczema), Baumer, et al., Inflamm. Allergy Drug Targets, 2007, Mar.; 6(1), 17-26 (abst.), guardedly report, "AWD 12-281 (GW 842470) is currently under clinical evaluation for the topical treatment of atopic dermatitis. Results concerning clinical efficacy of this potent and selective PDE4 inhibitor are anxiously awaited."

5. **Working Examples:** No examples show treatment of a claim 30 disorder. The sole biological data demonstrates only PDE4 inhibition, and does not indicate the PDE4 subtype tested. Applicants do not provide highly predictive competent evidence or recognized tests to treat all conditions recited for the claimed compounds.

   The compounds are disclosed to inhibit PDE-4 activity and the specification recites that these compounds therefore treat all diseases susceptible to amelioration by PDE-4 inhibition, including COPD, RA or IBD, diseases/conditions for which Applicants provide no competent evidence. Furthermore, Applicants have not provided competent evidence that the instantly disclosed tests are highly predictive for all uses disclosed and embraced by the claim language for the intended host.

6. **Skill of those in the art:** The specification indicates that these compounds are potent and selective inhibitors of (PDE4) and are thus useful in the treatment of bronchial asthma, COPD, allergic rhinitis, psoriasis, atopic eczema (atopic dermatitis). The concept that PDE-4 inhibitors could treat such pathological conditions/diseases generally is contrary to what is known about PDE-4 inhibitors. Some PDE4 inhibitors cause vasculitis (blood vessel inflammation), which has hindered PDE-4 inhibitor clinical investigation. Development of SCH-351591 halted because of acute and chronic vasculitis in small to medium sized arteries, and vasculitis was a significant problem with CI-1018 and Ariflo® (cilomilast). The PDE-4 inhibitor IC542 triggered a generalized inflammatory response with extensive neutrophil infiltration in the gastrointestinal tract, nearby mesentery and thymus.

The state of the art (e.g., the articles by Sasaki, the European Respiratory Society, Lipworth, Barnes, Dyke and Kawasaki, discussed in detail above) supports that successful amelioration of bronchial asthma, COPD, allergic rhinitis, psoriasis, atopic eczema (atopic dermatitis) with PDE4 inhibitors is a subject for further investigation. See the discussion of PDE-4 above.

The history of the actual effectiveness of PDE-4 inhibitors is very short. PDE-4 inhibitors have been investigated for disorders ranging from AD to COPD to depression to schizophrenia to chronic lymphocytic leukemia (CLL). Except in the area of asthma, such efforts have met with very little success. As of the time of filing, and indeed up to now, the FDA has not approved any PDE-4 inhibitor for treatment of any disorder. Extensive effort to get cilomilast and Daxas® (roflumilast) to be effective against COPD has been without success, evidence of the skill level in this art. Whether these claimed compounds affect the same isoenzymes as cilomilast and roflumilast is not described.

7. **Quantity of experimentation needed to make or use the invention.** Based on the disclosure's content, an undue burden would be placed on one skilled in pharmaceutical arts to make and use the invention, since the disclosure gives the skilled artisan inadequate guidance regarding pharmaceutical use, for reasons explained above. The state of the art indicates the requirement for undue experimentation. The ability of an agent that inhibits PDE-4 to ameliorate all diseases or conditions recited by the present claims remains open to further study and proof.

Substantiation of utility and its scope is required when utility is "speculative," "sufficiently unusual" or not provided. See *Ex parte Jovanovics, et al.*, 211 USPQ 907, 909 (BPAI 1981). Also, note *Hoffman v. Klaus*, 9 USPQ2d 1657 (BPAI 1988) and *Ex parte Powers*, 220 USPQ 924 (BPAI 1982) regarding types of testing needed to support *in vivo* uses. Applicants' attention is drawn to the Revised Interim Utility and Written Description Guidelines, at 66 FR 1092-1099 (2001), emphasizing that "a claimed invention must have a specific and substantial utility." See also MPEP 2163, *et. seq.* The disclosure in this application is not sufficient to enable the instantly claimed methods based solely on disclosure of inhibition of PDE-4 by compounds of Formula (I).

MPEP 2164.01(a) states,

> A conclusion of lack of enablement means that, based on the evidence regarding each of the above [Wand] factors, the specification, at the time the application was filed, would not have taught one skilled in the art how to make and/or use the full scope of the claimed invention without undue experimentation. *In re Wright*, 27 USPQ2d 1510, 1513 (Fed.Cir. 1993).

The above consideration clearly justifies that conclusion here and undue experimentation would be required to practice Applicants' invention. Consideration of the above factors demonstrates that this application does not sufficiently enable claims 17 and 18. In view of the pharmaceutical nature of the invention, the unpredictability of relationship between PDE-4 and specific diseases/conditions, one of ordinary skill in this art would have to undergo an undue amount of experimentation to use the instantly claimed invention commensurate in scope with claims 17 and 18.

Reply to Feb. 1, 2008 Comments

Applicant states that the data in Table 1, page 34, demonstrate that a selection of the claimed compounds inhibit PDE4 activity, and the rejection above indicated that claims so directed would be enabled by the specification. No such claims are pending.

Regarding Grootendorst (cited by Applicants), only a single PDE4 inhibitor, roflumilast, was tested and the researchers cautiously noted (p. 64, col. 1): "Those findings indicate that roflumilast might be indicated for the treatment of allergic rhinitis symptoms. However, whether PDE4 inhibitors really are an effective treatment for patients with allergic rhinitis should be evaluated in follow-up studies during the pollen season." Grootendorst also noted adverse effects of roflumilast in allergic rhinitis patients (p. 64, col. 2). Regarding COPD, Grootendorst reports testing with only a single PDE4 inhibitor, cilomilast.

Souness (cited by Applicants), in discussing the future of PDE4 inhibition therapy for COPD, suggests (p. 141, col. 2), "PDE4 inhibitors show efficacy in several preclinical models of airway inflammation, which suggest that they have potential in the treatment ... of ... COPD." Souness also reported (p. 145, col. 1) the failure of PDE4 therapy of COPD. "Zardaverine ... was inactive in patients with COPD." Ariflo was observed (p. 148, col. 2) to have adverse gastrointestinal side-effects and its effectiveness with asthma patients was reported (p. 149, col. 2) as "unimpressive."

Compare both Grootendorst and Souness with the more recent (2007) European Respiratory Society article, discussed in detail above, reporting that there is no current treatment for COPD and recommending prospective anti-inflammatory treatment.

No research has convincingly established cilomilast or roflumilast as effective against COPD, and they have not been approved for any disorder, despite much effort. No PDE4 inhibitors have been approved in the US (or Europe) for COPD, despite some early encouraging results. This shows that getting PDE4 inhibitors to be effective has proved very difficult. COPD is an exceptionally difficult disorder; in fact, at the present time, no drug has been found which alters the progression of the disease.

Rejections Under 35 USC 103

Claims 1-13, 15, 17 and 18 are rejected under 35 U.S.C. 103(a) as being obvious over Hatzelmann, et al., WO 2004018451, entitled to the filing date of 20030806.

Hatzelmann describes piperidinylpyridazinones as inhibitors of phosphor-diesterase PDE4 or PDE3/4 inhibitors. See the Hatzelmann compounds of Examples 4, 5, and 8-31. The claimed compounds are lower alkyl homologs of the Hatzelmann compounds, when the present compounds are further lower alkyl substituted on the 4-ring position of the 2H-pyridazine-3-one ring. Note that Hatzelmann (page 2, description of Formula 1 compounds) teaches that the 2H-pyridazine-3-one ring may be substituted in the 3- and 4-ring position by hydrogen or lower alkyl.

Presently claimed compounds that are lower alkyl homologs of Hatzel-mann would have been obvious to one of ordinary skill in the art at the time of the

present invention for the expected utility of the Hatzelmann compounds. One of ordinary skill in the art would have been motivated to prepare the claimed compounds as lower alkyl homologs of the Hatzelmann compounds, because such structurally related compounds are expected to possess similar properties. It has been held that compounds that are structurally homologous to prior art compounds are *prima facie* obvious, absent a showing of unexpected results.

> An obviousness rejection based on similarity in chemical structure and function entails the motivation of one skilled in the art to make a claimed compound, in the expectation that compounds similar in structure will have similar properties.

In re Payne, 203 USPQ 245, 254 (CCPA 1979). See also *In re Papesch*, 137 USPQ 43 (CCPA 1963) and *In re Dillon*, 16 USPQ2d 1897 (Fed.Cir. 1991) (discussed in MPEP § 2144) for an extensive case law review pertaining to obviousness based on close structural chemical compound similarity. See also MPEP § 2144.08, I[II.A.4(c). Compounds that are homologs (compounds differing by the successive addition of the same chemical group, e.g., by CH3-groups), as here, are generally of sufficiently close structural similarity that there is a presumed expectation that such compounds possess similar properties. *In re Wilder*, 195 USPQ 426 (CCPA 1977). Hatzelmann establishes a *prima facie* case of obviousness for the presently claimed compounds. Absent the presentation of verifiable data establishing the unobviousness of the claimed compounds over Hatzelmann, or other procedures as explained above, this rejection is deemed sound.

Hatzelmann has a common assignee and two common inventors with the instant application. Based upon the earlier effective filing date of Hatzelmann, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in Hatzelmann was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in Hatzelmann, prior to the effective filing date of Hatzelmann under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that this application and Hatzelmann are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). This rejection might also be overcome by showing that Hatzelmann is disqualified under 35 U.S.C. 103(c) as prior art in a rejection under 35 U.S.C. 103(a). See MPEP § 706.02(l)(1) and § 706.02(l)(2).

Reply to Feb. 1, 2008 Comments

At page 9, Applicants state: "Since the present application is commonly owned with the Hatzelmann, et al. application, this reference is disqualified as prior art under 35 U.S.C. 103(c)." This statement, without more, is insufficient to overcome this rejection. 35 U.S.C. 103(c)(1) reads: "Subject matter developed by another person, which qualifies as prior art only under ... subsection[s] (e) ... of section 102 of this title, shall not preclude patentability under this section where the subject matter and the claimed invention were, **at the time the claimed invention was made**, owned by the

same person or subject to an obligation of assignment to the same person." A statement of present common ownership is not sufficient. *In re Onda,* 229 USPQ 235 (Comm'r Pat. 1985). See also MPEP 706.02(l)(2). Establishing Common Ownership or Joint Research Agreement; II. Evidence Required To Establish Common Ownership.

Applications and references (whether patents, patent applications, patent application publications, etc.) will be considered by the examiner to be owned by, or subject to an obligation of assignment to the same person, at the time the invention was made, if the applicant(s) or an attorney or agent of record makes a statement to the effect that the application and the reference were, **at the time the invention was made**, owned by, or subject to an obligation of assignment to, the same person. **The statement concerning common ownership should be clear and conspicuous** (e.g., on a separate piece of paper or in a separately labeled section).

Conclusion

Applicant's amendment necessitated the new grounds of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the

shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Cecilia M. Jaisle, J.D. whose telephone number is 571-272-9931. The examiner can normally be reached on Monday through Friday; 8:30 am through 5:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James O. Wilson can be reached on 571-272-0661. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Cecilia M. Jaisle, J.D. 3/22/2008

/James O. Wilson/ Supervisory Patent Examiner, Art Unit 1624